



D-33-12-5-18 CT-5-RI-D-IV

REMEDIAL INVESTIGATION

VOLUME IV APPENDICES C THROUGH G

CALDWELL TRUCKING COMPANY SITE TOWNSHIP OF FAIRFIELD, NEW JERSEY

EPA WORK ASSIGNMENT
NUMBER 69-2LB3
CONTRACT NUMBER 68-01-6699

NUS PROJECT NUMBER S796

JANUARY 1986



Park West Two Cliff Mine Road Pittsburgh, PA 15275 412-788-1080

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SUBMITTED FOR NUS BY:

EONARD C. JOHNSON

PROJECT MANAGER

APPROVED:

DEBRA WROBLEWSKI REGIONAL MANAGER

REGIONAL M

001 097

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E	DOSE-RESPONSE EVALUATION
F	RESIDENTIAL WELL SURVEY RESULTS
G	SAMPLE CALCULATIONS

CTC 001 0975

APPENDIX C

SURFACE WATER INVESTIGATION ANALYTICAL RESULTS

TRAF	LE NUMBER FIC REPORT RIPTION:		CT-SM-000 BB422 BLANK	CT-SM-001 8B404 SM/SD-1	CT-SW-002 88405 SW/SD-5	CT-SM-003 BB406 SM/SD-6	CT-SM-003A BB413 DUP 003 SM/SD-6	CT-9M-004 88407 SM/SD-2	CT-SM-006 BB408 SM/SD-3	CT-SW-006 B8409 SW/SD-4
***	VOLATILES :	***			•					
PP	CAS NO	COMPOUND								
44V	76-09-2	METHYLENE CHLORIDE	48R	44 .	160	200	1718	67	1188	864
	67-64-1	ACETONE	ND	ND	ND	ND	ND	4,6J	10	11
	75-36-4	1, 1-0 I CHLOROETHENE	ND	ND	ND .	ND	ND	ND	ND	ND
30V	156-60-5	TRANS-1,2-DICHLOROETHENE	ND	NO	ND	ND	ND ND	11 ND	9 .	16
23V 11V	67-66-3 71-66-6	CHLOROFORM 1,1,1—TRICHLOROETHANE	ND ND	ND ND	ND ND	ND NO	ND '	NO	ND ND	12 ND
B7V	79-01-6	TRICHLOROETHENE	ND	ND	ND	7.4R	6.88R	248A	238A	628R
47	71-43-2	BENZENE	NO	ND	ND	ND	ND	ND	4J	ND
	127-18-4	TETRACHLOROETHENE	ND	ND	ND	ND	ND	9	43	6
PP	CAS NO	COMPOUND							٠	
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	16BR	138R	148R	ND	128A	ND	118A	ND
36B						***	ND	ND	ND	ND
	50-32-8	BENZO(A)PYRENE	ND	ND	ND	ND .	NU	, 	NU	NU
738		BENZO(A)PYRENE	ND	ND	ND	ND	NU		NU	NU
738	60-32-8	BENZO(A)PYRENE COMPOUND	ND	MD	ND	ND	NU		no	
738 *** PP	60-32-8 ACIDS *** CAS NO	COMPOUND	ND	ND	ND .	ND	NU		no	
738 PP	60-32-8 ACIDS *** CAS NO		ND	ND	ND	ND	NU			
738 PP 10 P	60-32-8 ACIDS *** CAS NO	COMPOUND FOR THIS CATEGORY	ND	ND	ND	ND	NU			

SAMPLE NUMBER : TRAFFIC REPORT NUMBER : DESCRIPTION:	CT~SW-000 88422 BLANK	CT-SM-001 88404 SM/SD-1	CT-SW-002 88405 SW/SD-5	CT-SM-003 BB406 SM/SD-6	CT-SW-003A 88413 DUP 003 SW/SD-6	CT-SW-004 , 88407 SW/SD-2	CT-SW-005 88408 SW/SD-3	CT-SW-008 88409 SW/90-4

1024-57-3 HEPTACHLOR EPOXIDE	.058	ND	ND	ND	ND	ND	ND	ND
1031-07-8 ENDOSUFAN SULFATE	. 27	ND	ND	ND	ND	ND	ND	ND

SAMPLE NUMBER: TRAFFIC REPORT NUMBER; DESCRIPTION:			EPORT NUMBER , MBB682 MBB654	CT-8M-002 MBB666 SM/SD-6	CT-SW-003 MBB666 SW/SD-6	CT-SW-003A MBB673 DUP 003 SW/SD-6	CT-SM-004 MBB667 SM/SD-2	CT-SM-006 M8B868 SM/SD-3	CT-SM-006 M88669 SM/SD-4	
***	NORGAN I CS	東東京			•					
PP	CAS NO	COMPOUND								
1		ALUMINUM	300	788	1766	952	627	312	60 1	478
2		ANTIMONY	NO	ND	NO	ND	ND	ND	208J	178J
3		ARSENIC	11	ND	ND	ND	. ND	ND	ND	ND
4		BARIUM	ND	ND	ND	[60]	- NO	ND	ND	ND
6		CADMIUM	ND	ND	ND	ND	ND	ND	19	12
7		CALCIUM	17490	29430	21210	28150	21750	42350	43340	47320
9		CHROMIUM	ND	ND	16	20	MD	ND	82	21
9		COBALT	NO	ND	ND	[21]	ND	ND	ND	ND
10		COPPER	19	20	41	ND	NO	[21]	46	66
11		IRON	133	5023	2773	1828	1476	2626	2307	2290
12		LEAD	7.2	14	20.0	16	18	26	12	11
13		MAGNESIUM	[3596]	9680	6364	6202	6469	12610	11080	11960
14		MANGANESE	ND	643	261	239	216	277	270	296
16		MERCURY	NO	ND	ND	.44	· ND	ND	ND	ND
16		NICKEL	[23]	[88]	49	[88]	MD	[22]	[28]	[86]
17		POTASSIUM	ND	[1008]	[1686]	[1652]	[1622]	[1092]	[1065]	[1110]
19		SILVER	ND	11J	MD	ND	ND	ND ·	49J	6 3 J
20		SODIUM	10120	17680	21060	22580	23100	28030	32600	34070
22		TIN	ND	ND	NO	ND	NO	ND	ND	[31]
23		VANADIUM	ND	[84]	[29]	[22]	ND	ND	ND	[21]
24		ZINC	106	63	8	29	29	82	60	66

TRAF	LE NUMBER : FIC REPORT (RIPTION:	NUMBER :	-	CT-SW-OOO M88582 BLANK	CT-8W-001 MBB664 SW/SD-1	CT-SM-002 MBB665 SM/SD-6	CT-SM-003 MBB666 SM/SD-6	CT-SM-003A MBB673 DUP 003 SW/SD-6	CT-SM-004 MBB667 SM/SD-2	CT-SM-006 MBB668 SM/SD-3	CT-SM-006 MBB669 SM/SD-4
***	GEOCHEMI CAL	PARAMETERS ***			•						
PP	CAS NO	COMPOUND									
		CYANIDE		ND	23.OR	23.OR	ND	ND	ND	ND	ND
		SULFATE .		4.0	12.6	16.0	4.0	8.0	6.0	2.5	61.0
		TDS		164.OR	800.0R	280.OR	294.OR	196.0R	326.OR	290.0R	318.OR
		CARBONATE		0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
		BICARBONATE		22.0	92.0	62 .0	58 .0	66.O	100.0	90.0	98.0
		COD		ND	60.0	27.0	20.0	16.0	22.0	27.0	22.0
		TOC		ND	20.0	11.0	11.0	11.0	11.0	8.0	8.0
		AMMON I A		ND	ND	ND	0.67	. 9	ND	. 34	ND
		TKN		ND	NO	ND	8.6	ND	1.0	ND	ND
		CHLORIDE		18.0	31.0	87.0	41.0	45.0	70.0	66.0	66.0
		tox		20.8J	96.6R	108R	74.6R	65.6R	61.8R	89.4R	76.6R

Notes:

- 1. ND, not detected.
- 2. Jr estimated value.
- 3. []; contaminant detected below contract detection limits, but greater than instrument detection limits.
- 4. B: contaminant detected in blank.
- 6. *: indicates duplicate analysis is not within control limits.
- 6. Ri data rejected.
- 7. DM: dats missing.
- 8. NA: not enalyzed.

Existing wells CTMM1, CTMM3, GH-1, GH-2, GH-2 and new wells P1, P1A, P2 and P2A were constructed with PVC screen and riser pipe.

New wells MM-1, 2, 3, 4, 5, 6, 7, 8, 9, 10 and exiting well CTBR were open barehole wells constructed using carbon steel surface casing.

Only new wells MM-1A, 2A, 3A, 4A, 6A, 6A were constructed with stainless steel screen and riser pipe

RIPTION	NUMBER .	CT-SN-007 B6410 SN/SD-7	CT-SN-0 88411 SM/80-8
VOLATILES	***		
CAS NO	COMPOUND		
		72 0	590
			ND:
	-		ND
			ND
			ND
	* *		NO ·
		• - •	NO
		- -	ND ND
BASE/NEUTR	ALS *** COMPOUND		
CAS NO			
	BIS(2-ETHYLHEXYL)PHTHALATE	ND	MD
	BIS(2-ETHYLHEXYL)PHTHALATE BENZO(A)PYRENE	ND 11	ND NO
117-81-7			
	75-09-2 67-64-1 75-36-4 166-60-5 67-66-3 71-55-6 79-01-6 71-43-2 127-18-4	75-09-2 METHYLENE CHLORIDE 67-64-1 ACETONE 75-35-4 1,1-DICHLOROETHENE 166-60-6 TRANS-1,2-DICHLOROETHENE	CAS NO COMPOUND 75-09-2 METHYLENE CHLORIDE 720 87-84-1 ACETONE ND 76-36-4 1,1-DICHLOROETHENE 2.6J 168-80-6 TRANS-1,2-DICHLOROETHENE 420 87-86-3 CHLOROFORM ND 71-56-6 1,1,1-TRICHLOROETHANE 520 79-01-6 TRICHLOROETHENE 340 71-43-2 BENZENE 38 127-18-4 TETRACHLOROETHENE 4J

GAMPI PE:

SAMPLE NUMBER: CT-SM-007 CT-SM-009
TRAFFIC REPORT NUMBER: BB410 BB411
DESCRIPTION: SM/SD-7 SM/SD-8

ND

. 11BR

1024-67-3 HEPTACHLOR EPOXIDE ND 1031-07-8 ENDOSUFAN SULFATE ND

SAMPLE NUMBER :	CT-SW-007	CT-8W-008
TRAFFIC REPORT NUMBER :	MB8670	MB8671
DESCRIPTION:	SM/SD-7	SW/SD-8
*** INORGANICS ***	•	

PP	CAS NO	COMPOUND		
1		ALUMINUM	7611	17790
2		ANT I MONY	ND '	ND
3		ARSENIC	22	16
4		BARIUM	[129]	[194]
6		CADMIUM	6.0	ND
7		CALCIUM	59090	40830
8		CHROMIUM	. 28	44
9		COBALT	ND	87
10		COPPER	26	47
11		IRON	9668	41940
12		LEAD	366	634
13		MAGNES I UM	17870	14 100
14		MANGANESE	1867	1114
15		MERCURY	NO	ND
16		NICKEL	43	. 63
17		POTASSIUM	[1197]	[1616]
19		SILVER	11J	ND
20		600 I UM	10490	15750
22		TIN	ND	ND
23		VANAD I UM	50	76
24		ZINC	99	218

SAMPLE NUMBER: TRAFFIC REPORT NUMBER: DESCRIPTION:

CAS NO

CT-SM-007 MBB670 SM/SD-7 CT-SW-008 MBB671

3D-7 SM/SD-8

*** GEOCHEMICAL PARAMETERS ***

CYANIDE	16.0R	16 . OR
SULFATE	65.0	2.5
TDS	274.OR	172.OR
CARBONATE	0.0	0.0
BICARBONATE	132.0	70.0
COO	48.0	104.0
TOC	4.4	4.8
AMMON I A	ND	. 9
TKN	. 78	2.0
CHLORIDE	28.0	20.0
TOX	267J	76.9R

Notes:

- 1. ND: not detected.
- 2. Jr estimated value.
- 3. []: contaminant detected below contract detection limits, but greater than instrument detection limits.
- 4. B: contaminant detected in blank.

COMPOUND

- 6. A: indicates duplicate analysis is not within control limits.
- 6. A: data rejected.
- 7. DM: data missing.
- 8. NA: not enelyzed.

Existing wells CTMM1, CTMM3, GH-1, GH-2, GH-2 and new wells P1, P1A, P2 and P2A were constructed with PVC screen and riser pipe.

New wells MM-1, 2, 3, 4, 5, 6, 7, 8, 9, 10 and exiting well CTBR were open borehole wells constructed using carbon steel surface casing.

Only new wells MM-1A, 2A, 3A, 4A, 5A, 6A were constructed with stainless steel screen and riser pipe

SAMP	PLE TYPE:	SD								
TRAF	LE NUMBER FIC REPORT RIPTION:		CT-SD-001 88414 SM/S0-1	CT-SD-002 BB415 SW/SD-6	CT-SD-003 BB4 16 SW/SD-6	CT-SD-003A 88423 SM/SD-6	CT-SD-004 88417 SW-SD-2	CT-SD-006 BB4 18 SW/SD-3	CT-SD-008 88419 SW/SD-4	CT-SD-007 BB420 SW/SD-7
***	VOLATILES	***								
PP	CAS NO	COMPOUND								
44V	76-09-2 67-64-1 75-15-0	METHYLENE CHLORIDE ACETONE CARBON DISULFIDE	378R 41R NO	40BR ND ND	4 19R ND NO	478R ND 43	aer 1aor ND	288A 91A ND	88A 79BR ND	30R 61R 2J
10V 30V	75-34-3 156-60-5	1,1-DICHLOROETHANE TRANS-1,2-DICHLOROETHENE	ND NO	NO NO	ND ND	NO NO	ND ND	ND ND	ND ND	ND 4J
23V	67-66-3	CHLOROFORM	ND	ND .	ND	ND	ND	ND	ND	2.J
14H	78-93-3	2-BUTANONE	ND	ND	NO	NO	NO	ND	ND	ND
117	71-66-6	1, 1, 1-TRICHLOROETHANE	ND	ND	ND	ND	NO	ND	ND	2 J
87V	79-01-6	TRICHLOROETHENE	ND	ND	ND	NO	ND	12	ND	2
4٧	71-43-2	BENZENE	ND	NO	NO	ND	ND	ND	ND	ND
95V	127-18-4	TETRACHLOROETHENE	ND	ND	ND	ND	ND	11	ND	ND
86V	108-88-3	TOLUENE	ND	ND	ND	NO	ND .	ND	ND	ND
***	BASE/NEUTR	ALS ***								
PP	CAS NO	COMPOUND								
	65-85-0	BENZOIC ACID	ND	ND ,	ND	ND	ND	ND	ND	ND
808	86-73-7	FLUORENE	ND	ND	NO	ND	ND	ND	ND	ND
818	85-01-8	PHENANTHRENE	ND	ND	ND	ND	ND	ND ·	ND	ND
788	120-12-7	ANTHRACENE	ND	ND	ND '	ND	NO	ND	ND	ND
68B	84-74-2	DI-N-BUTYL PHTHALATE	ND	ND	ND	ND	ND	ND	ND	ND
398	206-44-0	FLUORANTHENE	ND	ND	ND	NO	ND	ND	ND	ND
849	129-00-0	PYRENE	ND	ND	ND	ND	ND	ND	ND	ND
728	56-55-3	BENZO(A)ANTHRACENE	NO	ND	ND	ND	ND	ND	ND	ND
66B	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	1700J	ND ·	ND	ND	ND	ND	ND	ND
768	218-01-9	CHRYSENE	ND	ND	ND	ND	ND	ND	ND	ND
74B	205-99-2	BENZO(B&K)FLUORANTHENES	ND	ND	ND	ND	ND	ND	ND	ND

					•					
SAMP	LE TYPE:	SD					•	•		
	LE NUMBER		CT-SD-001 88414	CT-SD-002 884 16	CT-SD-003 884 16	CT-SD-003A 66423	CT~6D-004 88417	CT-SD-006 88418	CT-SD-006 88419	CT-SD-007 88420
	RIPTION:	TOWN TO THE THE TOWN TO THE TO	SM/80-1	SM/8D-6	SM/SD-6	SW/SD-6	SW-SD-2	SM/SD-3	SW/SD-4	SM/SD-7
738	60-32-8	BENZO(A)PYRENE	ND	ND	ND	ND ND	ND	ND	ND	ND
83B	193-39-6	INDENO(1,2,3-CD)PYRENE	ND	ND	ND	ND	ND	ND	NO	NO
798	191-24-2	BENZO(GHI)PERYLENE	ND	ND	ND	ND	ND	ND	NO	ND .
***	ACIDS ***									
PP	CAS NO	COMPOUND			•					
	•									
NO P	ARAMETERS I	FOR THIS CATEGORY								
***	PESTICIDES	***								
PP	CAS NO	COMPOUND								,
	72-66-9	4,4'-DDE	ND	ND	NO	ND	ND	ND	ND	ND
	72-54-8	4 , 4 1 -DDD	ND	ND	ND	ND	ND	ND	ND	ND .
	11097-69	AROCLOR-1254	ND	ND	NO ·	ND	ND	ND	ND	NO
	12672-29	AROCLOR-1248	ND	ND	ND	ND	ND	ND	ND	ND

SAMP	LE TYPE:	SD									
TRAF	LE NUMBER : FIC REPORT RIPTION;			CT-5D-001 MBB674 SW/SD-1	CT-SD-002 M88676 SM/SD-5	CT-SD-003 MBB676 SM/SD-6	CT-SD-003A MBB683 SW/SD-6	CT-SD-004 MBB677 SW-SD-2	CT-SD-006 MB9679 SM/SD-3	CT-SD-008 MBB679 SM/SD-4	CT-SD-007 M88600 SW/SD-7
***	NORGANICS	***			-						
PP	CAS NO	COMPOUND									
1		ALUMINUM		12000	8800	14000*	£100*	DM	12000	12000	14000
2		ANTIMONY	•	ND	NO	ND	ND	DM	ND	ND	ND
3		ARSENIC		ND	ND	ND	ND	DM	ND	ND .	ND
4		BARIUM		ND	ND	ND	ND	DM ·	ND	ND	ND
5		BERYLLIUM		ND	ND	ND	ND	DM	ND	ND :	ND
6		CADMIUM		ND	ND	7	6	DM	ND .	ND	ND
7		CALCIUM		[2000]	[1000]	5000	[4000]	DM	9200	[2000]	ND
8		CHROMIUM		20	10	70*	40*	DM	20	20	ND
9		COBALT		ND	ND	ND	ND	DM	ND	ND	ND
10		COPPER		60	20	150*	110*	DM	[10]	20	ND
11		IRON		19000	9900	17000*	12000*	DM4	2 3000	19000	18000
12		LEAD		110	2900	408*	120*	DM	6	27	79J
13		MAGNESIUM		ND	ND	ND	ND	DM	6000	ND	ND
14		MANGANESE		140	100	160*	92*	DM	420	260	260J
15		MERCURY		ND	. 16	. 2	ND	DM	ND	ND	ND
16		NICKEL		ND	ND	MD	ND	DM4	ND	ND	ND
17		POTASSIUM		ND	ND	ND	ND	DM4	[2000]	ND	ND
18		SELENIUM	•	ND	ND	ND	ND	DM	ND	ND	ND
19		SILVER		ND	ND	ND	ND	DM	ND	ND	ND
20		SOD I UM		ND	ND	ND	ND	DM	ND	ND	ND
. 21		THALLIUM		ND	ND	ND	ND	DM	ND	ND	ND
22		TIN		ND	ND	ND	ND	DM	ND	ND	ND
23	•	VANAD I UM		70	ND	70	[40]	·DM	60	40	40
24		ZINC		120	170	190*	120*	DM	60	60	110

SAMPLE TYPE.

SAMPLE NUMBER :

TRAFFIC REPORT NUMBER :

DESCRIPTION:

CT-80-001 MR8674 SW/SD~1

CT-60-002 M88676 SM/80-6

CT-80-003 MBB676 8M/SD-6

CT-6D-003A CT-6D-004 MBB683 MBB677 SW/SD-6 SM-SD-2

M88678 SW/9D~3

CT-60-006

CT-SD-006 CT-SD-007 MB8679 SM/SD-4

MARRES SW/SD-7

*** GEOCHEMICAL PARAMETERS ***

CAS NO

COMPOUND

NO PARAMETERS FOR THIS CATEGORY

Notes:

- 1. ND: not detected.
- 2. J: estimated value.
- 3. []: contaminant detected below contract detection limits, but greater than instrument detection limits.
- 4. B: contaminant detected in blank.
- 5. A: indicates duplicate analysis is not within control limits.
- 6. R. date rejected.
- 7. DM: data missing,
- 8. NA: not analyzed.

Existing wells CTMM1, CTMM3, GH-1, GH-2, GH-2 and new wells P1, P1A, P2 and P2A were constructed with PVC screen and riser pipe. New wells MM-1, 2, 3, 4, 6, 6, 7, 8, 9, 10 and exiting well CTSR were open borehole wells constructed using carbon steel surface casing. Only new wells MM-1A, 2A, 3A, 4A, 5A, 6A were constructed with steinless steel screen and riser pipe

SAMPLE NUMBER : TRAFFIC REPORT NUMBER : DESCRIPTION:			CT-SD-008 88421 SW/SD-8	CT-8D-107 BC369 SM/8D-8	CT-SD-108 BC360 SD-9	CT-80-108A BC364 80-8	CT-SD-101 BC361 SM/SD-4
***	VOLATILES	***					
PP	CAS NO	COMPOUND					
140	75-09-2	METHYLENE CHLORIDE	98A	178A	168R	2 18R	348A
	67-64-1	ACETONE	ND	298R	ND	ND	10J B R
	75-15-0	CARBON DISULFIDE	ND	MD	MD	ND	ND
OV	75-34-3	1,1-DICHLOROETHANE	ND	MD	9	5 J	ND
VO	156-60-5	TRANS-1, 2-DICHLOROETHENE	ND	ND	11	6)	ND
y E	67-86-3	CHLOROFORM	ND	ND	ND	ND	ND
4H	78-93-3	2-BUTANONE	ND	168A	ND	10JBR	10JBA
17	71-55-6	1, 1, 1~TRICHLOROETHANE	ND	ND	61	ND	ND
74	7 9- 01-6	TRICHLOROETHENE	ND	MD	29	24	ND
V	71-43-2	BENZENE	ND	ND .	ND	5JBR	6JBR
6V	127-18-4	TETRACHLOROETHENE	NO	ND	ND	NO	ND
6V	108-88-3	TOLUENE	ND	ND	12	27	ND
**	BASE/NEUTR	ALS ***					
PP	CAS NO	COMPOUND					
,	66-86-0	BENZOIC ACID	ND	ND	ND	2500J	ND
ОВ	86-73-7	FLUORENE	ND	ND	ND	500J	MD
18	85-01-8	PHENANTHRENE	ND	NO	600J	640	420J
88	120-12-7	ANTHRACENE	ND	NO	ND	600J	420J
88	84-74-2	DI-N-BUTYL PHTHALATE	ND	220JBR	800JBR	520 9 R	420JBR
98	206-44-0	FLUORANTHENE	ND	ND	600J	820	830
48	129-00-0	PYRENE	ND	ND	E003	ND	920
28	66-55-3	BENZO(A)ANTHRACENE	.ND	NO	ND	6001	490
	117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	ND	NO ·	800JR	860R	980R
68		DIDIE EINICHERICH IN INC.					
68	218-01-9	CHRYSENE	ND	ND	ND -	2001	670

U	PLE TYPE:	SD					
SAMPLE NUMBER : TRAFFIC REPORT NUMBER : DESCRIPTION:		CT-SD-008 BB421 SW/SD-8	CT-SD-107 BC369 SM/SD-9	CT~SD-108 BC360 SD~8	CT-SD-108A 8C364 SD-8	CT-SD-109 BC361 SM/SD-4	
738 838 798	60-32-8 193-39-6 191-24-2	BENZO(A)PYRENE INDENO(1,2,3-CD)PYRENE BENZO(GHI)PERYLENE	ND ND ND	ND ND ND	ND ND ND	ND ND	480J 420J
***	ACIDS ***						
PP	CAS NO	COMPOUND					
NO P	PARAMETERS I	FOR THIS CATEGORY					
RRR	PESTICIDES	ガネギ					
PP	CAS NO	COMPOUND					
	72-56-9	4,41-DDE	NO	230	16	21	6.8
	72-54-8 11097-69	4,4'-DDD AROCLOR-1264	ND 12268	160 4 100 J	63 ND	76 ND	11 ND

SAMPLE TYPE:

1

SAMPLE NUMBER :

TRAFFIC REPORT NUMBER :

DESCRIPTION:

CT-80-009 MB8681 SM/SD-8 CT-80-107 MBD601

SW/80-8

CT-8D-108 MBD602

80-08

CT-SD-108A CT-SD-109 MR8726 MBD603

8D-8

MBD603 8M/8D-4

*** GEOCHEMICAL PARAMETERS ***

PP CAS NO

COMPOUND

NO PARAMETERS FOR THIS CATEGORY

Notes

- 1. ND: not detected.
- 2. J. estimated value.
- 3. []: contaminant detected below contract detection limits, but greater than instrument detection limits.
- 4. Bi contaminant detected in blank.
- 5. *: indicates duplicate analysis is not within control limits.
- 6. R. data rejected.
- 7. DM: data missing.
- 9. NA: not analyzed.

Existing wells CTMN1, CTMN3, GH-1, GH-2, GH-3 and new wells P1, P1A, P2 and P2A were constructed with PVC screen and riser pipe.

New wells MN-1, 2, 3, 4, 6, 6, 7, 8, 9, 10 and exiting well CTBR were open borehole wells constructed using carbon steel surface casing.

Only new wells MN-1A, 2A, 3A, 4A, 5A, 6A were constructed with stainless steel screen and riser pipe

SAMPLE TYPE: SD							
SAMPLE NUMBER : TRAFFIC REPORT NUMBER : DESCRIPTION:			CT-SD-008 MB8691 SM/SD-8	CT-8D-107 MBD801 SM/SD-8	CT-8D-108 MBD602 SD-8	CT-SD-108A M88726 SD-8	CT-SD-109 MBD603 SM/SD-4
***	NORGANICS	共東京					
PP	CAB NO	COMPOUND					
1		ALUMINUM	7800	14900*J	7 620 *J	42 10≭J	12700 ≯ J
2		ANT I MONY	ND .	MD	ND	ND	ND
3		ARSENIC	ND	26	12	11	26
4		BARIUM	ND	[101]	[89]	[22]	[60]
5		BERYLLIUM	ND	ND	MD	[0.7]	[1.2]
6		CADMIUM	MD	6.9×j	MD	MD	5.1AJ
7		CALCIUM	ND	[1230]	[887]	[886]	ND
θ		CHROMIUM	ND	17	2 3 R	12R	24
9		COBALT	ND	[7.6]	[0.0]	[9.9]	[12]
10		COPPER	ND	NO	ND	ND	NO
11		IRON	22000	24000	9160	7110	19400
12		LEAD	180J	676	77	01	62
13		MAGNESIUM	ND	[2600]	[2170]JR	[1620]J	[2960]J
14		MANGANESE	1 2 0J	2261	76J	24RJ	4 12 J
16		MERCURY	ND	ND	MD	MO	ND
16		NICKEL	ND	[6.9]J	MD	[7.9]J	[14]J
17		POTASSIUM	ND	[981]J	[77 8]J	[463]J	[766]J
18		SELENIUM	ND	ND	ND	ND	ND
19		SILVER .	ND	ND	NO	ND	ND
20		900 (UM	ND	NO	ND	MD	ND
21		THALLIUM	ND	NO	ND	ND	[6.2]
22		TIN	ND	ND	ND	NO	ND
23		VANAD I UM	. 40	76	[24]	[21]	61
24		ZINC	76	161	96R	6 1R	105

D

CTC 001 0993

APPENDIX D
TOXICITY PROFILES

APPENDIX D TOXICITY PROFILES

Toxicity profiles are provided for indicator compound selected in Section 9.2.1. These profiles, where indicated, were prepared by Clement Associates.

D.1 Benzene

Health Effects

Benzene is a known human leukemogen. The International Agency for Research on Cancer (IARC) weight-of-evidence category is 1 (sufficient evidence in humans) (ICF, Inc., 1985). Following chronic exposure, leukemia is usually preceded by effects on the hepatic system (NAS, 1980). Mutagenic test systems using rats and other laboratory animals indicate toxic effect on bone marrow cells, including changes in chromosome number and chromosome breakage (NAS, 1980).

Reproductive effects have been demonstrated in rats and mice following oral and inhalation exposures (TD_{LO} 9 gm/kg to 12 gm/kg; TC_{LO} 17 ppm to 500 ppm) (USDHHS, 1985). Effects include post-implementation mortality, effects on fertility, fetotoxicity, and developmental abnormalities (USDHHS, 1985).

Acute exposure to benzene produces a wide range of signs and symptoms (NAS, 1980). Acute effects are transitory but may lead to lasting chronic effects such as anemia (NAS, 1980). Chronic exposure to benzene in humans produces toxic effects to the hematopoietic system (NAS, 1980). Reported effects include myelocytic anemia, thrombocytopenia, and leukopenia (NAS, 1980). Data are inadequate for deriving dose-response relationships (NAS, 1980).

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

The EC $_{50}$ values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range from 5,300 µg/liter to 386,000 µg/liter. However, only values for the rainbow trout (5,300 µg/liter) were obtained from a flow-through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate toxicity for relatively volatile compounds like benzene. A chronic test with <u>Daphnia magna</u> was incomplete, with no adverse effects observed at test concentrations as high as 98,000 µg/liter.

D.2 1,1,1-Trichloroethane

Health Effects (Clement Associates, 1985)

1,1,1-Trichloroethane (1,1,1-TCA) was retested for carcinogenicity because in a previous study by NCI (1977), early lethality precluded assessment of carcinogenicity. Preliminary results indicate that 1,1,1-TCA increased the incidence of combined hepatocellular carcinomas and adenomas in female mice when administered by gavage (NTP, 1984). There is evidence that 1,1,1-trichloroethane is mutagenic in <u>Salmonella typhimurium</u> and causes transformation in cultured rat embryo cells (USEPA, 1980). These data suggest that the chemical may be carcinogenic.

Other toxic effects of 1,1,1-TCA are seen only at concentrations well above those likely in an open environment. The most notable toxic effects of 1,1,1-trichloroethane in humans is anesthesia at very high concentrations and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above); cardiovascular effects, including premature ventricular contractions, decreased blood pressure, and sensitization to epinephrine-induced arrhythmia; and adverse effects on the lungs, liver, and kidneys. Irritation of the

skin and mucous membranes resulting from exposure to 1,1,1-TCA has also been reported. The oral LD $_{50}$ value of 1,1,1-trichloroethane in rats is about 11,000 mg/kg.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

The acute toxicity of 1,1,1-trichloroethane to aquatic species is rather low, with the LC_{50} concentration for the most sensitive species tested being 52.8 mg/l. No chronic toxicity studies have been done on 1,1,1-trichloroethane, but acute-chronic ratios for the other chlorinated ethanes ranged from 2.8 to 8.7. 1,1,1-Trichloroethane was only slightly bioaccumulated with a steady-state bioconcentration factor of nine and an elimination half-life of two days.

No information on the toxicity of 1,1,1-trichloroethane to terrestrial wildlife or domestic animals was available in the literature reviewed.

D.3 Chloroform

Health Effects

Chloroform is a suspected carcinogen. The IARC weight-of-evidence category is 2B (sufficient evidence in animals) (ICF, Inc., 1985). No definitive conclusions can be reached concerning mutagenicity; however, there is some indication (from binding studies and from mutagenicity tests that utilize endogenous or in vivo metabolism) that chloroform may be a weak mutagen (USEPA, 1984a).

Chloroform appears to have teratogenic potential in laboratory animals when inhaled (USEPA, 1984a). Embryotoxic and teratogenic effects have been noted at 100 ppm (USEPA, 1984a). When administered orally, toxic effects on fetal development occur only at maternally toxic levels (USEPA, 1984a).

Acute toxicity to chloroform to experimental animals and humans is characterized by neurological, renal, hepatic, and cardiac effects (Sittig, 1985; USEPA, 1984a).

The lowest reported lethal dose to humans (LD_{LO}) is 140 mg/kg (USDHHS, 1985). Acute inhalation of 100 ppm is sufficient to produce hepatic effects in mice (NAS, 1977).

Chronic exposure in humans has been shown to produce renal and cardiac effects (USEPA, 1984a). Gastrointestinal and behavioral changes were noted in humans exposed via inhalation for 1 year; the TC_{LO} is 1,000 mg/m³ (USDHHS, 1985). Chronic toxicity experiments with experimental animals did not establish a noeffect for systemic toxicity (USDHHS, 1985).

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Limited information is available concerning the toxicity of chloroform to organisms exposed at known concentrations. Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 μ g/liter. Twenty-seven day LC₅₀ values of 2,030 and 1,240 μ g/liter were reported for embryo-larval tests with rainbow trout in water at two levels of hardness.

An equilibrium bioconcentration factor of six with a tissue half-life of less than 1 day was determined for the bluegill. Although chloroform is not strongly bioaccumulated, it is thought to be widely distributed in the environment and can be detected in fish, water birds, marine mammals and various crops.

D.4 <u>1,1-Dichloroethene</u>

Health Effects (Clement Associates, 1985)

1,1-Dichloroethene (VDC) caused kidney tumors in males and leukemia in one study on mice exposed by inhalation, gave equivocal results in other inhalation studies, but gave negative results in rats and mice following oral exposure and in hamsters following inhalation exposure. VDC was mutagenic in several bacterial assays for genetic toxicity. 1,1-Dichloroethene did not appear to be teratogenic but did cause embryotoxicity and fetotoxicity when administered to rats and rabbits by

inhalation. Chronic exposure to oral doses of VDC as low as 5 mg/kg/day caused liver changes in rats. Acute exposure to high doses causes central nervous system depression, but neurotoxicity has not been associated with low-level chronic exposure. The oral LD $_{50}$ value for the rat is 1,500 mg/kg and for the mouse it is 200 mg/kg.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

1,1-Dichloroethene was not very toxic to freshwater or saltwater species, with acute LC_{50} values generally being in the range of 80 to 200 mg/liter. A chronic study in which no adverse effects were observed indicated that the acute-chronic ratio was less than 40; a 13 day study which produced an LC_{50} of 29 mg/liter indicated that the acute-chronic ratio is greater than 4.

No reports of the toxicity of 1,1-dichloroethene to terrestrial wildlife or domestic animals were found in the literature reviewed.

D.5 <u>1,2-Trans-Dichloroethene</u>

Health Effects (Clement Associates, 1985)

Very little information concerning exposure only to 1,2-trans-dichloroethene (1,2-trans-DCE) is available. There are no reports of carcinogenic or teratogenic activity by 1,2-trans-DCE in animals or humans. It is reportedly nonmutagenic in a variety of test systems. Like other members of the chlorinated ethylene series, 1,2-trans-DCE has anesthetic properties. Exposure to high vapor concentrations has been found to cause nausea, vomiting, weakness, tremor, and cramps in humans. Repeated exposure via inhalation of 800 mg/m³ (8 hours/day, 5 days/week, for 16 weeks) was reported to produce fatty degeneration of the liver in rats. The intraperitoneal injection LD₅₀ value for the rat is 7,536 mg/kg.

Although nephrotoxic and cardiac sensitizing effects are associated with exposure to 1,1-dichloroethene, the 1,2-DCE isomers have not been investigated with

respect to this type of effects. 1,2-trans-Dichloroethene can inhibit aminopyrine demethylation in rat liver microsomes in vitro, and it may thus interact with the hepatic drug-metabolizing monooxygenase system.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Practically no information concerning the toxicity of 1,2-trans-DCE to wildlife and domestic animals exists. The reported 96-hour LC50 value under static conditions is 135,000 μ g/liter for the bluegill. Under the same test conditions, the LC50 value for 1,1-dichloroethene is 73,900 μ g/liter. Recommended criteria for protection of aquatic life are based primarily on data concerning 1,1-dichloroethene.

D.6 Ethylbenzene

Health Effects (Clement Associates, 1985)

Ethylbenzene has been selected by the National Toxicology Program to be tested for possible carcinogenicity, although negative results were obtained in mutagenicity assays in <u>Salmonella typhimurium</u> and <u>Saccharomyces cerevisiae</u>. There is recent animal evidence that ethylbenzene causes adverse reproductive effects. Ethylbenzene is a skin irritant, and its vapor is irritating to the eyes at a concentration of 200 ppm (870 mg/m³) and above. When experimental animals were exposed to ethylbenzene by inhalation, 7 hours/day for 6 months, adverse effects were produced at concentrations of 600 ppm (2,610 mg/m³) and above, but not at 400 ppm (1,740 mg/m³). At 600 ppm rats and guinea pigs showed slight changes in liver weight, and monkeys and rabbits experienced histopathologic changes in the testes. Similar effects on the liver and kidney were observed in rats fed ethylbenzene at 408 and 680 mg/kg/day for 6 months.

Ethylbenzene was acutely toxic to freshwater species at levels greater than 32 mg/liter. No chronic toxicity was reported, but the highest test dose (440 µg/liter) was only one-hundredth of the 96-hour LC₅₀ for the particular species being tested. No studies on the bioaccumulation of ethylbenzene were reported in the information reviewed, but a bioconcentration factor of 95 was calculated using the log octanol/water partition coefficient. No information on the toxicity of ethylbenzene to domestic animals and terrestrial wildlife was found in the sources reviewed.

D.7 Methylene Chloride

Health Effects (Clement Associates, 1985)

Methylene chloride is currently under review by the National Toxicology Program (NTP 1984). Preliminary results indicate that it produced an increased incidence of lung and liver tumors in mice and mammary tumors in female and male rats. In a chronic inhalation study, male rats exhibited an increased incidence of sarcomas in the ventral neck region (Burek et al. 1984). However, the authors suggested that the relevance and toxicological significance of this finding were uncertain in light of available toxicity data. Methylene chloride is reported to be mutagenic in bacterial test systems. It also has produced positive results in the Fischer rat embryo cell transformation test. However, it has been suggested that the observed cell-transforming capability may have been due to impurities in the test material. There is no conclusive evidence that methylene chloride can produce teratogenic effects.

In humans, direct contact with methylene chloride produces eye, respiratory passage, and skin irritation. Mild poisonings due to inhalation exposure produce somnolence, lassitude, numbness and tingling of the limbs, anorexia, and lightheadedness, followed by rapid and complete recovery. More severe poisonings generally involve correspondingly greater disturbances of the central and

peripheral nervous systems. Methylene chloride also has acute toxic effects on the heart, including the induction of arrhythmia. Fatalities reportedly due to methylene chloride exposure have been attributed to cardiac injury and heart failure. Methylene chloride is metabolized to carbon monoxide in vivo, and levels of carboxyhemoglobin in the blood are elevated after acute exposure. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and distal paresis. An oral LD50 value of 2,136 mg/kg, and an inhalation LC50 value of 88,000 mg/m³/30 min are reported for the rat.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Very little information concerning the toxicity of methylene chloride to domestic animals and wildlife exists. Acute values for the freshwater species <u>Daphnia magna</u>, the fathead minnow, and the bluegill are 224,000, 193,000, and 224,000 μ g/liter, respectively. Acute values for the saltwater species, mysid shrimp and sheepshead minnow, are 256,000 and 331,000 μ g/liter, respectively. No data concerning chronic toxicity are available. The 96-hour EC₅₀ values for both freshwater and saltwater algae are greater than the highest test concentration, 662,000 μ g/liter.

D.8 Tetrachloroethene

Health Effects (Clement Associates, 1985)

Tetrachloroethene wsa found to produce liver cancer in male and female mice when administered orally by gavage (NCI 1977). Unpublished gavage studies in rats and mice performed by the National Toxicology Program (NTP) showed hepatocellular carcinomas in mice and a slight, statistically insignificant increase in a rare type of kidney tumor. NTP is also conducting an inhalation carcinogenicity study. Elevated mutagenic activity was found in Salmonella strains treated with tetrachloroethene. Delayed ossification of skull bones and sternebrae were reported in offspring of pregnant mice exposed to 2,000 mg/m³ of tetrachloroethene for 7 hours/day on days 6-15 of gestation. Increased fetal

resorptions were observed after exposure of pregnant rats to tetrachloroethylene. Renal toxicity and hepatoxicity have been noted following chronic inhalating exposure of rats to tetrachloroethene levels of 1,356 mg/m³. During the first 2 weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m³) of tetrachloroethene produced signs of central nervous system depression, and cholinergic stimulation was observed among rabbits, monkeys, rats, and guinea pigs.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Tetrachloroethene is the most toxic of the chloroethenes to aquatic organisms but is only moderately toxic relative to other types of compounds. The limited acute toxicity data indicated that the LC $_{50}$ value for saltwater and freshwater species were similar, around 10,000 μ g/liter; the trout was the most sensitive (LC $_{50}$ = 4,800 μ g/liter). Chronic values were 840 and 450 μ g/liter for freshwater and saltwater species respectively, and an acute-chronic ratio of 19 was calculated.

No information on the toxicity of tetrachloroethene to terrestrial wildlife or domestic animals was available in the literature reviewed.

D.9 Toluene

Health Effects (Clement Associates, 1985)

There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans. The National Toxicological Program is currently conducting an inhalation carcinogenicity bioassay in rats and mice.

Oral administration of toluene at doses as low as 260 mg/kg produced a significant increase in embryonic lethality in mice. Decreased fetal weight was observed at doses as low as 434 mg/kg, and an increased incidence of cleft palate was seen at

doses as low as 867 mg/kg. However, other researchers have reported that toluene is embryotoxic but not teratogenic in laboratory animals. There are no accounts of a teratogenic effect in humans being linked to toluene exposure.

Acute exposure to toluene at concentrations of $375\text{--}1,500~\text{mg/m}^3$ produces central nervous system depression and narcosis in humans. However, even exposures to quantities sufficient to produce unconsciousness fail to produce residual organ damage. The rat oral LD50 value and inhalation LCLO value are 5,000 mg/kg and 15,000 mg/m³, respectively. Chronic inhalation exposure to toluene at relatively high concentrations produces cerebellar degeneration and an irreversible encephalopathy in mammals.

Toluene, in sufficient amounts, appears to have the potential to significantly alter the metabolism and resulting bioactivity of certain chemicals. For example, coadministrations of toluene along with benzene or styrene has been shown to suppress metabolism of the benzene or styrene in rats. The estimated weighted average bioconcentration factor for toluene and the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is calculated to be 10.7.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Of five freshwater species acutely tested with toluene, the cladoceran <u>Daphnia magna</u> was most resistant. The EC₅₀ and LC₅₀ values for all species range from 12,700 to 313,000 μ g/liter. No chronic tests are available for freshwater species. The two freshwater algal species tested are relatively insensitive to toluene with EC₅₀ values of 245,000 μ g/liter or greater being reported.

D.10 Trichloroethene

Health Effects (Clement Associates, 1985)

Trichloroethene is carcinogenic to mice after oral administration, producing hepatocellular carcinomas (NCI, 1976; NTP, 1982). It was found to be mutagenic using several microbial assay systems. Trichloroethene does not appear to cause reproductive toxicity or teratogenicity. TCE has been shown to cause renal toxicity, hepatotoxicity, neurotoxicity, and dermatological reactions in animals following chronic exposure to levels greater than 2,000 mg/m 3 for 6 months. Trichloroethylene has low acute toxicity; the acute oral LD $_{50}$ value in several species ranged from 6,000 to 7,000 mg/kg.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

There was only limited data on the toxicity of trichloroethene to aquatic organisms. The acute toxicity to freshwater species was similar in the three species tested, with LC_{50} values of about 50 mg/liter. No chronic toxicity tests were reported.

No information on the toxicity of trichloroethene to domestic animals or terrestrial wildlife was available in the literature reviewed.

D.11 Xylenes

Health Effects (Clement Associates, 1985)

The National Toxicology Program (NTP) is testing xylene for carcinogenicity by administering it orally to rats and mice. Although the results have not been finalized, it does not appear to be carcinogenic in rats. Results have not been reported for mice. Xylene was found not to be mutagenic in a battery of short-term assays. Xylene was not teratogenic but has caused fetotoxicity in rats and mice. Acute exposure to rather high levels of xylene affects the central nervous

system and irritates the mucous membranes. There is limited evidence of effects on other organ systems, but it was not possible to attribute these effects solely to xylene as other solvents were present. The oral LD $_{50}$ value of xylene in rats was 5,000 mg/kg.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Xylene adversely affected adult trout at concentrations as low as 3.6 mg/liter in a continuous flow system and trout fry avoided xylene at concentrations greater than 0.1 mg/liter. The LC50 value in adult trout was determined to be 13.5 mg/liter. LC50 values for other freshwater fish were around 30 mg/liter in a static system, which probably underestimated toxicity. Only a few studies have been done on the toxicity of xylene to saltwater species. These indicated that the m- and o-xylene isomers probably have similar toxicities and are probably less toxic than p-xylene, and that saltwater species are generally more susceptible than freshwater species to the detrimental effects of xylene (LC50 = 10 mg/liter for m- and o-xylene and LC50 = 2 mg/liter for p-xylene). However, it should be stressed that these generalizations are based on results from limited data.

No information on the toxicity of xylenes to terrestrial wildlife and domestic animals was available in the literature reviewed. However, because of the low acute toxicity of xylenes it is unlikely that they would be toxic to wild or domestic birds and mammals.

D.12 Acetone

Health Effects

Acetone is considered to have a low toxicity. Acute exposure to high concentrations via inhalation in humans has been demonstrated to produce respiratory, sensory, metabolism, and central nervous system (CNS) effects at concentrations ranging from 182 ppm to 12,000 ppm (USDHHS, 1985).

Few data are available on the effects of chronic exposure to acetone in humans or experimental animals. Workers exposed to 1,000 ppm (3 hours/day) for 7 to 15 years had symptoms of respiratory tract irritation, dizziness, and weakness (Clayton and Clayton, 1981).

Toxicity to Wild Life and Domestic Animals

Acetone is considered to have a low toxicity to aquatic organisms (Clement Associates, 1985).

D.13 Polychlorinated Biphenyls (PCBs)

Health Effects (Clement Associates, 1985)

In humans exposed to PCBs (in the workplace or via accidental contamination of food), reported adverse effects include chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs have been reported in rhesus monkeys at the lowest dose level tested, 11 µg/kg body weight/day over a period of several months.

Toxicity to Wildlife and Domestic Animals (Clement Associates, 1985)

Polychlorinated biphenyls are bioaccumulated and can be biomagnified. Therefore, their toxicity increases with length of exposure and position of the exposed species on the food chain. The toxicity of the various PCB mixtures is also dependent on their composition. Because of the complexity of PCB toxicity, only general effects will be discussed here.

The 96-hour LC $_{50}$ values for rainbow trout, bluegills, and channel catfish were around 20 mg/liter. The same species exposed for 10 to 20 days had LC $_{50}$ values of about 0.1 mg/liter. Invertebrate species were also adversely affected, with some species having 7-day LC $_{50}$ values as low as 1 µg/liter. In general, juvenile organisms appeared more susceptible to the effects of PCBs than either eggs or adults.

Three primary ways in which PCBs can affect terrestrial wildlife are outright mortality, adversely affecting reproduction, and changing behavior. greater than 200 ppm in the diet or 10 mg/kg body weight (bw) caused some mortality in sensitive bird species exposed for several days. Doses around 1,500 ppm (diet) or about 100 mg/kg (bw) caused extensive mortality in these sensitive species. They generally caused some mortality in all species, with the level being dependent on the length of exposure and the particular PCB mixture. Some mammalian species are especially susceptible to PCBs. For example, mink died when fed as little as 5 ppm in the die (equivalent to less than 1 mg/kg bw/day). PCBs caused lower egg production; deformities; decreased hatchability, growth, and survival; and some eggshell thinning in reproductive studies on chickens fed doses of 20 ppm in the diet (1 mg/kg bw). Mink fed 1 ppm in the diet (0.2 mg/kg bw) had lower reproductive success, and there are indications that an increased incidence of premature births in some marine animals was linked to PCB exposure. Behavioral effects on wildlife include increased activity, decreased avoidance response, and decreased nesting, all of which could significantly influence survival in the wild.

No toxic effects on domestic animals other than chickens were reported in the sources reviewed, but susceptible species would probably be affected in a similar manner to laboratory animals and wildlife.

D.14 Arsenic

Health Effects

Arsenic is a known human carcinogen (USEPA, 1985a). The IARC weight of evidence category is 1 (sufficient evidence in humans). In humans, exposure to arsenic is associated with tumors of the skin, kidneys, genital organs, and visual organs (USEPA, 1985a). Arsenic has been shown to be mutagenic in several test systems and to induce chromosomal aberrations in in vivo and in vitro systems (USEPA, 1985a).

Arsenite and arsenate can cross the placenta in mammals (USEPA, 1985a). Sodium arsenate and arsenite have embryolethal effects and teratogenic potential in several mammalian species (USEPA, 1985a).

Effects associated with acute oral exposure to arsenic in humans and experimental animals include severe gastrointestinal damage (leading to shock, coma, and death), muscular cramps, facial edema, and cardiovascular reactions (USEPA, 1984b). Acute toxicity varies with the valence form of the element; trivalent arsenic is approximately four times more toxic than pentavalent arsenic (USEPA, 1984b). Effects associated with acute inhalation exposure to high concentrations include irritant effects, peripheral nervous system disturbances, and reversible effects on the hepatic system.

Chronic exposure to arsenic compounds has been shown to produce toxic effects, such as systemic irreversible damage (USEPA, 1985a). Effects noted in chronic animal studies include body weight changes, decreased blood hemoglobin, hepatic damage, and kidney damage (USEPA, 1985a). Peripheral and CNS effects have been observed in humans and experimental animals (USEPA, 1984b).

Toxicity to Wildlife and Domestic Animals

Although arsenic is concentrated in aquatic organisms, it is not bioconcentrated along the food chain. The EC₅₀/LC₅₀ values for arsenic range from 812 μ g/l to 41,760 μ g/l (trivalent inorganic arsenic and 7,400 to 10,800 μ g/l pentavalent inorganic arsenic (USEPA, 1980a). A life cycle test with <u>Daphmia magna</u> exposed to sodium arsenate resulted in a chronic value of 912 μ g/l (USEPA, 1980a).

D.15 Cadmium

Health Effects

Cadmium is a suspected carcinogen. The IARC weight of evidence category is 2B (sufficient evidence in animals) (USEPA, 1985a). Cadmium and cadmium compounds have been shown to induce sarcomas or lung tumors when administered to animals via parenteral or inhalation exposure (USEPA, 1985a). There is some evidence of carcinogenicity in humans via inhalation exposure (USEPA, 1985a). There is no direct association between carcinogenicity and ingestion of cadmium in experimental animals or humans (USEPA, 1985a). Gene mutation studies in mammalian cell cultures, rec-assays in bacteria, chromosomal nondysfunction studies in intact animals, and other indicators of mutagenic damage indicate cadmium is mutagenic (USEPA, 1984c).

Experimental evidence indicates cadmium is teratogenic in animals (Kirsch-Volders, 1984). No observations of reproductive effects have been observed in humans (Kirsch-Volders, 1984).

Acute exposure to cadmium in experimental animals and humans results in renal dysfunction, hypertension, anemia, and altered liver microsomal activity (USEPA, 1985a). Chronic exposure to cadmium in experimental animals and humans results in renal dysfunction, hypertension, anemia, and altered liver microsomal activity (USEPA, 1985a). The critical target organ in humans chronically exposed to cadmium via ingestion is the kidney (USEPA, 1985a).

Toxicity to Wildlife and Domestic Animals

Fish and several invertebrates are sensitive to low levels of cadmium in water. Freshwater acute toxicity values range from 1 to 73,500 μ g/l for fish species and 3.5 to 28,000 μ g/l for invertebrate species (USEPA, 1980b). Salmonids and cladocerans are the most sensitive organisms (USEPA, 1980b).

D.16 Lead

Health Effects

Lead acetate and lead phosphate are suspected human carcinogens. The IARC weight-of-evidence category is 3 (cannot be classified as to its carcinogenicity) (USEPA, 1985a). Carcinogenic activity has been associated with ingestion of high doses in rats and some cases of renal tumors in long-term exposure to humans in the lead industry (USEPA, 1985a). Insufficient data are available on the mutagenicity of lead. Although lead has been shown to affect DNA synthesis, cell proliferation and in vivo DNA synthesis. Negative results have been obtained in all test systems utilized to assess its capacity to induce gene mutations in prokaryotes and eukaryotes (Kirsch-Volder, 1984).

Fetotoxic effects have occurred in experimental animals exposed to 600 to 800 ppm of lead through ingestion (USEPA, 1984d). Adverse effects have been noted in experimental animals at 5 to 10 ppm via drinking water and 10 mg/m 3 via inhalation (USEPA, 1984d). Teratogenic effects in experimental animals have been noted only when the maternal dose is injected (USEPA, 1984d).

Acute lead poisoning is rare (NAS, 1977). One child was reported to have consumed 1 g of lead per day before death (NAS, 1977). Oral doses of 300 mg/kg have been reported to be lethal to dogs (NAS, 1982). Chronic lead toxicity is severe and occurs even with low daily intake because of its accumulation in bone and tissue (Nas, 1977). Chronic exposure results in adverse effects on three target systems: the heme-hemoprotein system, the kidneys, and the central nervous system

(NAS, 1982). Disturbance in heme synthesis is considered to be the most critical effect (NAS, 1977). Infants and children represent a sensitive subpopulation with regard to lead exposure. Of major concern are the reported subtle effects of lead on behavior (NAS, 1982).

Toxicity to Wildlife and Domestic Animals

Acute toxicity data for lead for freshwater fish and invertebrate species indicate effects at concentration ranges of 124 to 542,000 μ g/l (USEPA, 1980d). Chronic toxicity values range from 12 to 174 μ g/l (USEPA, 1980d). Bioconcentration factors are 42 to 1,700 (USEPA, 1980d).

D.17 Nickel

Health Effects

Nickel is a known human carcinogen. The IARC weight-of-evidence category is 2A (limited evidence in humans) (USEPA, 1985a). Nickel has not been shown to be carcinogenic in experimental animals or humans following oral exposure (USEPA, 1985a). Increased incidences of respiratory tract cancers have been demonstrated in nickel refinery workers (USEPA, 1985a). Metallic nickel, nickel subsulfide, and nickel carbonyl have been shown to provide tumors in experimental animals via inhalation or intravenous injection. Nickel chloride has been shown to be negative for mutagenicity in <u>Eschericha coli</u> and <u>Bacillus subtilis</u> (USEPA, 1985a). Nickel chloride and nickel sulfate have been shown to be mutagenic in eukaryotic mutation test systems (USEPA, 1985a).

Nickel appears to be teratogenic in experimental animals (Kirsch-Volder, 1984). Adverse effects include fetal and postnatal mortality and developmental abnormalities (Kirsh-Volder, 1984).

Nickel and nickel salts are considered to have a low oral toxicity in experimental animals (NAS, 1977). Nickel carbonyl is extremely toxic to the lungs in humans

following acute inhalation (NAS, 1977). Few data are available on the effects of chronic exposure to nickel. Subchronic (6 weeks) oral exposure in laboratory animals to high concentrations is associated with depressed weight gain, alterations in hematology parameters, and cytochrome oxidase activity (USEPA, 1985a).

Toxicity to Wildlife and Domestic Animals

Acute toxicity values range from 510 μ g/l for <u>Daphnia magna</u> to 46,200 μ g/l for banded killfish (USEPA, 1980e). A relationship between nickel toxicity and water hardness is demonstrated in several species. Chronic aquatic toxicity values range from 14.8 μ g/l to 530 μ g/l (USEPA, 1980e).

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APPENDIX E DOSE-RESPONSE EVALUATION

TABLE E-1

DOSE-RESPONSE EVALUATION HUMAN HEALTH EFFECTS CALDWELL TRUCKING COMPANY SITE

		Unit Cancer Risk	Maximum Contaminant	Ambient Wa	ter Quality Criteria	Suggested No.	Acceptable
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	Ingestion of Biota(4)	Ingestion of Water(5)	Adverse Response Level (ug/l)(5)	
P.P. No.	Compound	-			·		
Volatile O	ganics		•				
4V	benzene	$2.9 \times 10^{-2} (W)$	0(b) 5(c)	0 (40 ug/i) ⁽¹⁾	0 (0.67 ug/I) ^(f)	10-day: 230 chronic: 70	200
6V	carbon tetráchloride	1.3 x 10 ⁻¹	-	0 (6.94 ug/l)(f)	0 (0.42 ug/l) ^(f)	1-day: 200 10-day: 20	~
7 V	chlorobenzene	-	-	-	488 ug/l	-	1.0
10V	1,2-dichloroethane	6.9 x 10 ⁻²	0(b) 1 (c)	0 (243 ug/l) ^(f)	0 (0.94 ug/I) ^(f)	-	-
11V	1,1,1-trichloroethane	1.6 x 10 ⁻³	0.20(b)(c)	1.03 g/l	19 mg/l	chronic: 1,000	38
13V	1, 1-dichloroethane	-	-		-	- ,	8.1
16V	chloroethane	-	-		-	-	~
23V	chloroform	7.0 x 10 ⁻²	-	0 (15.7 ug/l) ^(f)	0 (0.19 ug/l)	-	~
29V	1, 1-dichloroethene	1.47 x 10 ⁻¹	0.007(b)(c)	. 0 (1.85 ug/l)	0 (33 ng/l)	1-day: 1,000 chronic: 70	~
30V	1,2-trans-dichloroethene	-	-	-	~	1-day: 2,700 10-day: 270	~
38V	ethylbenzene	-	-	3.28 mg/l	2.4 mg/l	-	9.5

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE TWO

		Unit Cancer Risk	Maximum Contaminant	Ambient Wat	er Quality Criteria	Suggested No	Acceptable
Dose-Response Parameter P.P. No. Compound		Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	Ingestion of Biota ⁽⁴⁾	Ingestion of Water(5)	Adverse Respon Level (ug/l)(5)	se Daily Intake
44V	methylene chloride	— 1.4 × 10 ⁻² (I) 7.5 × 10 ⁻³ (O)	-	0 (15.7 ug/l)(f)(g)	0 (0.19 ug/l) ^(†) (9)	1-day: 13,0 10-day: 1,30 chronic: 150	
48V	bromodichloromethane	-	-	0 (15.7 ug/l)(f)(g)	0 (0.19 ug/l) ^(f) (9)	, -	0.039
85V	tetrachloroethene	6.0 x 10 ⁻²	-	0 (8.85 ug/I) ^(f) (9)	0 (0.88 ug/l) ^(f)	1-day: 2,30 10-day: 175 chronic: 20) <u> </u>
86V	toluene	-		424 mg/l	15 mg/l	1-day: 21,5 10-day: 2,20 chronic: 340	
87V	trichloroethene	1.2 x 10 ⁻²	0(b) 5(c)	0 (80.7 ug/l) ^(f)	0 (2.8 ug/l) ^(f)	1-day: 2,00 10-day: 200 chronic: 75	0 -
88V	vinyl chloride	1.75 x 10 ⁻² (!)	0(p)	0 (525 ug/l) ^(f)	0 (2.0 ug/l) ^(f)	-	-
13H	acetone	<u>-</u>	-	-	-	-	200
14H	2-butanone	-	- ,	-	-	10-day: 7,50 chronic: 750	1.4
15H	carbon disulfida	·	-	· <u>-</u>	-	-	-
16H	2-hexanone	-	_	-	<u>-</u>	-	-

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE THREE

		Unit Cancer Risk	Maximum Contaminant	Ambient Wa	ter Quality Criteria	Suggested No.	Acceptable
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	Ingestion of Biota(4)	Ingestion of Water(5)	Adverse Response Level (ug/l)(5)	Daily Intake (mg/day)(6)
P.P. No.	Compound						
17H	4-methyl-2-pentanone	-	-	-	-	-	7.3
20H	xylenes	•	-	-	-	1-day: 12,000 10-day: 1,200 chronic: 620	160
Base/Neut	ral Extractables						
18	acenaphthene	~	-	-	20 ug/l	-	-
88	1,2,4-trichlorobenzene	~	-	-	-	-	1.8
258	1,2-dichlorobenzene	~	-	2.6 mg/l	470 ug/l	-	6.3
26B	1,3-dichlorobenzene	~	.	2.6 mg/l	470 ug/l	-	6.3
27B	1,4-dichlorobenzene	~	-	2.6 mg/i	470 ug/ì	· · · · · · · · · · · · · · · · · · ·	7.5
39B	flouranthene	~	<u>-</u>	54 ug/l	188 ug/l		0.42
55B	naphthalene	~	_	-	-	-	18
62B	n-nitrosodiphenylamine	4.92 x 10 ⁻³	-	0 (16.1 ug/l)(f)	0 (7.0 ug/l) ^(f)	-	-
66B	bis(2-ethylhexyl)phthalate		-	50 mg/l	21 mg/l	-	42
67B	butyl benzyl phthalate	-	-		-	<u>.</u>	-

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE FOUR

		Unit Cancer Risk	Maximum Contaminant	Ambient Wat	er Quality Criteria	_ Suggested No.	Acceptable
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/i)(2)(3)	ingestion of Biota(4)	Ingestion of Water(5)	Adverse Response Level (ug/l)(5)	Daily intake (mg/day)(6)
P.P. No.	Compound						
688	di-n-butyl phthalate	-	-	154 mg/l	44 mg/l	-	88
69B	di-n-octyl phthalate	-	-		-	-	-
708	diethyl phthalate	-	-	1.8 g/i	434 mg/l	-	880
71B	dimethyl phthalate	-	-	2.9 g/l	350 mg/l	-	700
72B	benzo(a)anthracene	.	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	-
73B	benzo(a)pyrene	. 11.5	<u>-</u>	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l)(f)(h)	-	- -
748	benzo(b)fluoranthene	-	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	-
75B	benzo(k)fluoranthene	-	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	- .
768	chrysene	-	-	0 (31.1 ng/l) ^(f) (h)	0 (2.8 ng/l) ^{(f)(h)}	-	-
77B	acenaphthylene	-	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	· -
78B	anthracene	-		0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	-
79B	benzo(ghi)perylene	_	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}		- .
80B	fluorene	<u>-</u>	_	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l) ^{(f)(h)}	-	-
81B	phenanthrene	- ' ,	-	0 (31.1 ng/I)(f)(h)	0 (2.8 ng/l)(f)(h)	<u> </u>	-

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE FIVE

		Unit Cancer Risk	Maximum Contaminant	Ambient Wat	er Quality Criteria	_ Suggested No.	Acceptable
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	ingestion of Biota(4)	ingestion of Water(5)	Adverse Response Level (ug/l) ⁽⁵⁾	Daily Intake (mg/day)(6)
P.P. No.	Compound						
83B	indeno(1,2,3-cd)pyrene	· •	-	0 (31.1 ng/l)(f)(h)	0 (2.8 ng/l)(f)(h)	-	-
84B	pyrene	-	-	-	-	- .	· -
6Н	benzyl alcohol	-	-	-	· -	-	
8H	4-chloroaniline	- ·	-	-	-	-	· _ ·
8H	dibenzofuran		-	-	-	-	· -
9н	2-methylnaphthalene	-	-			-	-
Acid Extra	ctables	•				•	
65A	phenol	-	-	-	3.5 mg/l	-	7.0
1H	benzoic acid	-	-	-	-	- ·	. <u>-</u> :
2H	2-methyl phenol	- -	<u>-</u>	-	-	-	-
3Н	4-methyl phenol	<u>.</u>	_	- '	-	-	-
Pesticides.	/PCBs						
89P	aldrin	11.4	-	0 (0.079 ng/l) ^(f)	0 (1.2 ng/i) ^(f)	-	-
92P	4,4'-DDT	0.34	-	0 (0.024 ng/l) ^(f)	0 (>1.2 ng/l) ^(f)	· _	-

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE SIX

		Unit Cancer Risk	Maximum Contaminant	Amhient Wate	er Quality Criteria	Suggested No.	Acceptable
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	Ingestion of Biota ⁽⁴⁾	Ingestion of Water(5)	Adverse Response Level (ug/l) ⁽⁵⁾	Daily Intake (mg/day)(6)
P.P. No.	Compound				· · · · · · · · · · · · · · · · · · ·		
93P	4,4'-DDE	- ,	-	-	-	-	-
94P	4,4'-DDD	-	-	. -	-	-	-
95P	endosulfan I	-	-	159 ug/l	138 ug/l	-	0.28
97P	endosulfan sulfate	-	-	-	-	-	-
100P	heptachlor	3.37	-	0 (0.29 ng/l)(f)	0 (11 ng/i) ^(f)	-	-
101P	heptachlor epoxide	-	_	- .	- .	-	-
103P	beta-BHC	1.84	-	0 (54.7 ng/l) ^(f)	0 (23.2 ng/l) ^(f)	-	-
105P	gamma-BHC	1.33	4 ug/I(d)	0 (62.5 ng/l) ^(f)	0 (26.4 ng/l) ^(f)	-	-
106P	PCB-1242	4.34	-	0 (0.79 ng/l) ^(†)	0 (>12.6 ng/l) ^(f)	1-day: 125 10-day: 12.5	-
107P	PCB-1254	4.34	-	0 (0.79 ng/l) ^(†)	0 (>12.6 ng/l) ^(f)	1-day: 125 10-day: 12.5	-
110P	PCB-1248	4.34	-	0 (0.79 ng/l) ^(f)	0 (>12.6 ng/l) ^(f)	1-day: 125 10-day: 12.5	
111P	PCB-1260	4.34		0 (0.79 ng/l) ^(f)	0 (>12.8 ng/l) ^(f)	1-day: 125 10-day: 12.5	. -

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE SEVEN

		•	Unit Cancer Risk	Maximum Contaminant	Ambient Wat	er Quality Criteria
		Dose-Response Parameter	Slope Factor (mg/kg-day-1) ⁽¹⁾ (a)	Level (ug/i)(2)(3)	Ingestion of Biota ⁽⁴⁾	Ingestion of Water(5)
	P.P. No.	Compound				
	112P	PCB-1016	4.34	-	0 (0.79 ng/l) ^(f)	0 (>12.6 ng/l) ^(f)
	Inorganics					
		aluminum	_	_	45 ug/l	146 ug/l
		antimony	-	-	45,000 ug/l	-
		arsenic	15(H)	50(d)	0 (17.5 ng/l) ^(f)	0 (2.5 ng/l) ^(f)
		barium	-	1,000 ^(d)	-	.
		beryllium	2.6	-	0 (64.1 ng/l) ^(f)	0 (3.9 ng/l) ^(f)
		cadmium	7.8(H)	₁₀ (d)	· —	10 ug/l
	· .	chromium	41(W)	50(d)		CrVI – 50 ug/l CrIII – 179 mg/l
	•	cobalt .	-	-	- ·	-
		copper		1,000 ^(d)	_	1 mg/ (i)
		iron		300(d)	-	<u>-</u>
1054	CTC 001	lead	-	₅₀ (d)	-	50 ug/l

TABLE E-1
DOSE-RESPONSE EVALUATION
HUMAN HEALTH EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE EIGHT

		Unit Cancer Risk	Maximum Contaminant	Ambient Wat	er Quality Criteria
	Dose-Response Parameter	Slope Factor (mg/kg-day-1)(1)(a)	Level (ug/l)(2)(3)	Ingestion of Biota(4)	Ingestion of Water(5)
P.P. No.	Compound		,		
	magnesium	-	-	-	-
	maganese	-	50(e)	-	-
	mercury	· ·	2(d)	146 ng/i	10 ug/l
	nickel	1.15(W)	-	100 ug/i	15.4 ug/l
	tin	-	-	-	-
	silver	-	50	- ,	50
	vanadium	-	-	-	-
	zinc	-	5,000(e)	- .	5 mg/ (i)

CLC 001 1052

Notes:

- (a) The Unit Cancer Risk Slope Factor is calculated based on studies of oral exposure in animals, except for those indicated by "I" (animal inhalation), "W" (human occupational exposure), and "H" (human drinking water exposure).
- (b) Recommended Maximum Contaminant Level (RMCL).
- (c) Proposed Maximum Contaminant Level (PMCL).

TABLE E-1 **DOSE-RESPONSE EVALUATION HUMAN HEALTH EFFECTS CALDWELL TRUCKING COMPANY SITE PAGE NINE**

(d)	National Interim Primary Drinking Water Standard Maximum Contaminant Level (MCL).
(e)	Secondary Drinking Water Standard Maximum Contaminant Level (MCL).
(f)	The Ambient Water Quality Criteria (AWQC) for the maximum protection of human health is zero. Because zero may not attainable, the values tabulated correspond to a 10^{-6} carcinogenic risk.
(g)	Ambient Water Quality Criteria for halomethanes.
(h)	Ambient Water Quality Criteria for polycyclic aromatic hydrocarbons.
(i)	Organoleptic limit.
	Signifies criterion is not available or not applicable.
Sources:	
(1)	USEPA. 1985a.

- (1) (2) USEPA, 1982.
- (3) USEPA, 1985b.
- (4) USEPA, 1980.
- (5) USEPA, 1985c.
- (6) USEPA, 1984a.

TABLE E-2

DOSE-RESPONSE EVALUATION ENVIRONMENTAL EFFECTS CALDWELL TRUCKING COMPANY SITE

Compound	Ambient Water Quality Criteria Acute Effects (ug/l)(1)(2)	Ambient Water Quality Criteria Chronic Effects (µg/l)(1)(2)
methylene chloride	11,000	NR
acetone	NR	NR ·
1,1-dichloroethene	11,600	NR
trans-1,2-dichloroethene	NR	NR
chloroform	28,900	1,240
1,1,1-trichloroethene	NR	NR
trichloroethene	45,000	21,900
benzene	5,300	NR
tetrachloroethene	5,280	840
acenaphthene	1,700	NR
benzo (a)pyrene	NR	NR
arsenic	trivalent inorganic arsenic - 140(b) pentavalent inorganic arsenic - 850	trivalent inorganic arsenic – 72 ^(c)
barium	NR	NR

TABLE E-2
DOSE-RESPONSE EVALUATION
ENVIRONMENTAL EFFECTS
CALDWELL TRUCKING COMPANY SITE
PAGE TWO

Compound	Ambient Water Quality Criteria Acute Effects (µg/I)(1)(2)	Ambient Water Quality Criteria Chronic Effects (µg/l)(1)(2)
cadmium(a)	6.6	6.6
chromium(a)	CrVI - 11(b) CrIII - 2,024(b)	CrVI - 7.2(c) CrIII - 97.6(c)
lead(a)	99.9(b)	3.9(c)
nickei ^(a)	2,376(d)	NR
silver(a)	7.20 ^(d)	0.12
vanadium	NR	NR

Notes:

NR - Not Reported

- (a) Calculated based on a hardness of 139.6 mg/l as CaCO₃. Hardness was calculated using calcium and magnesium concentrations in surface water samples.
- (b) Maximum concentrations in a 30-day period.
- (c) Average concentration in a 30-day period.
- (d) Instantaneous maximum concentration.

Sources:

- (1) USEPA, 1980
- (2) USEPA, 1984b

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USEPA, June 1985c. <u>Guidance on Feasibility Studies under CERCLA</u>. Office of Emergency Remedial Response and Office of Waste Programs Enforcement, Washington, D.C.

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<u>Organic Chemicals</u>. Federal Register, Vol. 50, No. 219.

APPENDIX F RESIDENTIAL WELL SURVEY RESULTS

Address	Name	Well Depth (Feet)	Groundwater Use (1)	Comments
18 Carlos Drive	Roe	40	D, B, C, GW	
24 Carlos Drive (2)	Kleine	Approx. 12	D, B, C, GW	
25 Carlos Drive	Alcide	35	GW	Municipal Water
26 Carlos Drive	Habermas	65	D, B, C, GW	
31 Carlos Drive	Cuozzo	55	D, B, C, GW	
34 Carlos Drive	Doncoes		D, B, C, GW	
36 Carlos Drive (3)	Merano	75 - 77	D, B, C, GW	
44 Carlos Drive	Rumore		I, GW, Car washing	Municipal Water
45 Carlos Drive	Priore		GW, Occasional drinking	Municipal Water
46 Carlos Drive	Papera	20	D, B, C, GW	·
55 Carlos Drive	Bellino		D, B, C, GW	
2 Colt Street	Klein-Corallo	122	D, B, C, GW,	
12 Colt Street (4)	Capko	100	Occasional D, GW Carwashing	
42 Colt Street	Jernark		D, B, C	
5 Daniel Road East				Municipal Water
10 Dey Avenue	Sabanosh	125	D, B, C	
10 Glen Avenue	Cobb	70	D, B, C, GW	
15 Glen Avenue	Larson	30	B, GW, Swimming	Water was used for drinking until recently
20 Glen Avenue	Aksynowicz	50	D, B, C, GW	
34 Glen Avenue	Dowiak	57	D, B, C, GW	***
44 Glen Avenue	Slintak			Municipal Water
56 Glen Avenue	Napholz			Municipal Water
164 Little Falls Road	Chantivani			Municipal Water
	(Pennini – tenant)			•
176 Little Falls Road	Webb			Municipal Water
205 Little Falls Road	Roome and Lepore		В, С	Municipal Water
244 Little Falls Road (5)	Talamini	46	I, D, B, C, GW	·
248 Little Falls Road	Talamini	50	I, D, B, C, GW	
13 Maplewood Avenue	Mustardo		D, B, C, GW	
15 Maplewood Avenue	Kakuk	60	GW	Municipal Water

TABLE F-1
RESIDENTIAL WELL SURVEY RESULTS
CALDWELL TRUCKING COMPANY SITE
PAGE TWO

Address	Name	Well Depth (Feet)	Groundwater Use (1)	Comments
70 New Dutch Lane	The Singer Company			Municipal Water
9 Orlando Drive	Forth		GW	Municipal Water
3 Orlando Drive	Appaluccio	Shallow		Municipal Water
5 Orlando Drive	Russoniello			Municipal Water
6 Orlando Drive	Napolitano	, 		Municipal Water
224 Passaic Drive	Heisler Machine and Tool Co.	200 - 250	Cooling Extruded Material	Municipal Water
333 Passaic Drive	C.E. Conover and Company			Municipal Water
10 Pier Lane	Evans		B, GW	Municipal Water
24 Pier Lane	Rigan, Inc.	· .		Municipal Water
36 and 40 Pier Lane	Croft Realty			Municipal Water
66 Pier Lane (6)	Lurie	Approx. 80	GW	Municipal Water
69 Pier Lane	Lombardi	18 - 25	GW	
70 Pier Lane	Martin	41	GW	Municipal Water
102 Pier Lane	Greer			Municipal Water
114 Pier Lane	Himes		GW	Municipal Water
(Well location 110-108 Pier L	_ane)		•	
108 Pier Lane	Brisacher			Municipal Water
8 Shire Avenue	Morgan		D, B, C, GW	
18 Summit Avenue	Kakuk	60	D, B, C, GW	
20 Summit Avenue	Wallace	75	D, B, C, GW	
26 Summit Avenue	Pierce	72	D, B, C, GW	
35 Summit Avenue	Hooyman		D, B, C, GW	
7 Toll Terrace	Smith	60		Municipal Water
18 Toll Terrace*	Stanley	51	I, D, B, C, GW	
18 Toll Terrace (7)*	Stanley	51	GW, Carwashing	
10 Van Ness Avenue	Zavolda	225	D, B, C	
11 Van Ness Avenue (8)	Moldoch	120	D, B, C, GW	~ -
15 Van Ness Avenue	Capozzi		I, D, B, C, GW	

TABLE F-1
RESIDENTIAL WELL SURVEY RESULTS
CALDWELL TRUCKING COMPANY SITE
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Address	Name	Well Depth (Feet)	GroundwaterUse (1)	Comments
21 Van Ness Avenue	Crossley	Approx. 60	D, B, C, GW	
27 Van Ness Avenue	Serpico	20	GW	Municipal Water
33 Van Ness Avenue	Koch	80	D, B, C, GW	
38 Van Ness Avenue	Passafaro	85	D, B, C, GW	
45 Van Ness Avenue	Smith	65	D. B. C	
54 Van Ness Avenue	Grieco	Approx. 175	D, B, C, GW, Swimming	
58 Van Ness Avenue	Lovas	220	D, B, C, GW	

Notes:

- (1) D = Drinking
 - B = Bathing/Washing
 - C = Cooking
 - 1 = Irrigation
 - **GW = Garden Watering**
- (2) Well Sampled; CT-RW-003
- (3) Well Sampled; CT-RW-004
- (4) Well Sampled; CT-RW-005
- (5) Well Sampled; CT-RW-007
- (6) Well Sampled; CT-RW-001
- (7) Well Sampled; CT-RW-002
- (8) Well Sampled; CT-RW-006

 * = Two questionnaires submitted.
- -- = Signifies information was not provided.

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APPENDIX G
SAMPLE CALCULATIONS

APPENDIX G SAMPLE CALCULATIONS

G.1 Estimation of Contaminant Concentrations in the Passaic River

To estimate potential concentrations of contaminants in the Passaic River as impacted from the groundwater contaminant plume in the area, data from the piezometers located 250 feet (P-1) and 750 feet (P-2) from the river were used. River data obtained during the RI were also used. The following site-specific data was provided:

- River Elevation: 160.96 ft; river high because of rain
- P-1 Elevation: 161.63 ft; distance from river = 250 ft
- P-2 Elevation: 162.07; distance from river = 750 ft
- Hydraulic conductivity of the aquifer (permeability) = 1.82×10^{-4} cm/sec (average K, or glacial sediment) (3.86 gpd/ft²)

The following equations were used to calculate potential concentrations in the river:

$$Q_G = \frac{K}{2L} (h_1^2 - h_2^2) \cdot I \quad (Dupuit's Formula)$$
 (1)

$$M_{G} = Q_{G} \cdot \bar{c}/K' \tag{2}$$

$$C_{River} = \frac{M_G}{Q_G + Q_{River}}$$
 (3)

where: Q_G = flow from aquifer into the river (gpd)

K = hydraulic conductivity (3.86 gpd/ft²)

1 = distance between h_1 and h_2 (ft)

h₁ = head of groundwater (ft)

h₂ = head of river (assumed 160 ft)

= interface length between river and aquifer (2,200 ft)

M_G = mass loading from the aquifer (lb/day)

= average concentration of pollutant (lb/gal) (highest concentration of P-1 or P-2)

R = Retardation constant; assumed to be 1 (worst-case) (accounts for adsorption, degradation, dispersion, etc.)

QRiver = River flow (1) = 911 cfs - 5.89 x 10⁸ gpd (911 cfs is a mean value for water year 1981 - lower bound).

River flow (2) = 34 cfs = 2.2 x 10⁷ gpd (34 cfs is a minimum value for water year 1981 corresponding to worst-case scenario).

CRiver = diluted pollutant concentration in the river (lb/gal)

The highest pollutant concentration present in P-1 and P-2 from both rounds of sampling was chosen and carried through the calculation. A range was generated using minimum flow conditions and average flow conditions for the river. The final concentrations in the river were obtained by averaging the results from each piezometer to accurately characterize the nonuniform plume.

Example 1: For trichloroethene (TCE), the highest concentration is 7,200 μ g/l at P-2 (second round of sampling).

From Eq. (1)
$$-Q_G = \frac{3.86}{2(750)}(162^2 - 160^2) \cdot 2,200$$

= 3,646 gpd

From Eq. (2)
$$- M_G = (3.646) (6.01 \times 10^{-5})/1$$

= 0.22 lb/day

^{*} From USGS Water Year 1981 New Jersey Water Resources Data, Passaic River at Little Falls, New Jersey Station.

From Eq. (3):

(1) Low Flow
$$C_{River} = \frac{0.22}{3,646 + 2.2 \times 10^7}$$

= 1 x 10⁻⁸ lb/gal
= 1.2 µg/l

(2) Average Flow
$$C_{River} = \frac{0.22}{3,646 + 5.89 \times 10^8}$$

= 3.735 x 10⁻¹⁰ lb/gal
= 0.045 µg/l

Example: For 1,1-dichloroethene, the highest concentration is 250 μ g/l at P-1 (second round of sampling).

From Eq. (1)
$$-Q_G = \frac{3.86}{2(250)} (162^2 - 160^2) \cdot 2,200$$

= 10,938 gpd
From Eq. (2) $-M_G = (10,938) (2.086 \times 10^{-6})/1$

= 0.023 lb/day

From Eq. (3):

(1) Low Flow
$$C_{River} = \frac{0.023}{10,938 + 2.2 \times 10^7}$$

= 1.037 x 10⁻⁹ lb/gal
= 0.124 µg/l

(2) Average Flow
$$C_{River} = \frac{0.023}{10,938 + 5.89 \times 10^8}$$

= 3.905 x 10⁻¹¹ lb/gal
= 0.005 µg/l

G.2 Dose Estimates

A dose is defined as the amount of a compound (mg) absorbed by a receptor on a daily basis per kilogram of body weight. Doses can be calculated for lifetime or less than lifetime exposures. A dose can be estimated as follows:

Carcinogenic risk can be calculated as follows: ,

$$Risk = (q) (dose)$$

Sample calculations and site-specific assumptions used to estimate doses in Section 9.4.4 are provided in the following subsections.

G.2.1 Ingestion of Drinking Water

Assumptions used to estimate the dose associated with long-term ingestion of contaminated drinking water include:

- A receptor ingests of 2 liters of water per day.
- An average man weights 70 kg.
- 100 percent of the compound is absorbed in the gastrointestinal tract.

A dose associated with ingestion of the maximum concentration of TCE in RW-001 (8,600 μ g/I) can be estimated as follows:

Dose =
$$\frac{8,600 \text{ µg/l} \times 2 \text{ l/day} \times 1 \text{ mg/10}^3 \text{ ug}}{70 \text{ kg}} = 0.24 \text{ mg/kg-day}$$

Risk = (q)*(dose)

where:
$$q^* = 1.2 \times 10^{-2} \text{ (mg/kg-day)}^{-1} \text{ (Table E-1)}$$

Risk = $[1.2 \times 10^{-2} \text{ (mg/kg-day)}^{-1}][0.24 \text{ mg/kg-day}] = 2.9 \times 10^{-3}$

G.2.2 Inhalation During Showering

Assumptions used to estimate the dose associated with inhalation during showering include:

- 180 liters of water are used during showering (USEPA, 1985).
- 50 percent of the compound volatilizes to the air (Andelman, 1984).
- 0.33 hr/day are spent in the bathroom (USEPA, 1985).
- 1.2 m³/hr are inhaled (Andelman, 1985).
- The estimated dimension of a bathroom is 12 m³ (USEPA, 1985).
- 100 percent of a compound is absorbed upon entering the lungs.
- The weight of an adult is 70 kg.

A dose associated with inhalation of TCE in RW-001 (8,600 μ g/I) during showering can be calculated as follows:

190 I x
$$0.5$$
 x $8,600 \mu g/I = 817,000 \mu g$

$$\frac{817,000 \text{ ug}}{12 \text{ m}^3} = 68,083 \text{ ug/m}^3$$

Dose =
$$\frac{68,038 \text{ ug/m}^3 \text{ x } 1.2 \text{ m}^3/\text{hr x } 0.33 \text{ hr/day x } 1 \text{ mg/}10^3 \text{ ug}}{70 \text{ kg}}$$

= 0.38 mg/kg-day

Risk =
$$(q)*(dose)$$

where:
$$g^* = 1.2 \times 10^{-2} (mg/kg-day)^{-1} (Table E-2)$$

Risk =
$$[1.2 \times 10^{-2} (mg/kg-day)^{-1}][0.38 mg/kg-day] = 4.6 \times 10^{-3}$$

G.2.3 Dermal Exposure to Contaminated Surface Soil

Assumptions used to estimate the dose associated with dermal contact include:

- A lifetime soil accumulation ranges from 7,900 g to 110,000 g (Schaum, 1984).
- To assume a "worst-case" exposure, 100 percent of a compound is absorbed through the skin (McLaughlin, 1984). Ten percent of the pesticides are absorbed through the skin (McLaughlin, 1984).
- The weight of an adult is 70 kg.
- An expected lifetime is 70 years.

A dose associated with dermal contact with the maximum concentration of PCB-1248 (76,000 μ g/kg) detected in onsite surface soil can be calculated as follows:

Maximum Lifetime Soil Accumulation

Dose = $\frac{76,000 \text{ } \mu\text{g/kg} \times 7,900 \text{ g} \times 1 \text{ } \text{mg/}10^3 \text{ } \text{ug} \times 1 \text{ kg/}10^3 \text{ } \text{mg}}{70 \text{ kg} \times 70 \text{ years and } 365 \text{ days/year}}$

= 3.3 x 10^{-4} mg/kg-day

Risk = $(q)^*$ (dose)

where: $q^* = 4.34 (mg/kg-day)^{-1} (Table E-1)$

Risk = $[4.34 (mg/kg-day)^{-1}][3.3 \times 10^{-4} mg/kg-day] = 1.4 \times 10^{-3}$

Maximum Lifetime Soil Accumulation

Dose =
$$\frac{76,000 \text{ } \mu\text{g/kg} \text{ x } 110,000 \text{ g x 1 mg/}10^3 \text{ ug x 1 kg/}10^3 \text{ mg}}{70 \text{ kg x 70 years and 365 days/year}}$$

$$=$$
 4.7 x 10^{-3} mg/kg-day

where:
$$q^* = 4.34 (mg/kg-day)^{-1} (Table E-1)$$

Risk =
$$[4.34 (mg/kg-day)^{-1}][4.7 \times 10^{-3} mg/kg-day] = 2.0 \times 10^{-2}$$

G.2.4 Accidental Ingestion of Contaminated Surface Soil

Assumptions used to estimate the dose associated with accidental ingestion of contaminated soil include:

- 0.1 to 5 g of soil are ingested per day (Schaum, 1984).
- The "worst-case" exposure duration is 1,830 days (Schaum, 1984).
- The weight of a child (2 to 6 years old) likely to ingest soil is 14 kg (Schaum, 1984).
- An expected lifetime is 70 years.

A dose associated with ingestion of the maximum concentration of PCB-1248 (76,000 μ g/kg) detected in onsite surface soil can be calculated as follows:

Minimum Soil Ingestion Rate

Dose =
$$\frac{76,000 \text{ } \mu\text{g/kg} \times 0.1 \text{ } \text{g/day} \times 1,830 \text{ } \text{days} \times 1 \text{ } \text{mg/}10^3 \text{ } \text{ug} \times 1 \text{ } \text{kg/}10^3 \text{ } \text{g}}{14 \text{ } \text{kg} \times 70 \text{ } \text{years} \text{ } \text{and} \text{ } 365 \text{ } \text{days/year}}$$

$$=$$
 3.9 x 10^{-5} mg/kg-day

Risk =
$$(q)*(dose)$$

where:
$$q^* = 4.34 (mg/kg-day)^{-1} (Table E-1)$$

Risk =
$$[4.34 (mg/kg-day)^{-1}][3.9 \times 10^{-5} mg/kg-day] = 1.7 \times 10^{-4}$$

Maximum Soil Ingestion Rate

Dose =
$$\frac{76,000 \text{ µg/kg} \times 5 \text{ g/day} \times 1,830 \text{ days} \times 1 \text{ mg/}10^3 \text{ µg} \times 1 \text{ kg/}10^3 \text{ mg}}{14 \text{ kg} \times 70 \text{ years and 365 days/year}}$$

 $= 1.9 \times 10^{-3} \text{ mg/kg-day}$

Risk = (q)*(dose)

where: $q^* = 4.34 \text{ (mg/kg-day)}^{-1} \text{ (Table E-1)}$

Risk = $[4.34 (mg/kg-day)^{-1}][1.9 \times 10^{-3} mg/kg-day] = 8.4 \times 10^{-3}$

G.2.5 Dermal Exposure During Swimming

Assumptions used to estimate the dose associated with dermal exposure during swimming include:

- The body surface area of a child is 7,700 cm² (Versar, 1984).
- The average body weight of a child is 10 kg (Versar, 1984).
- Water flux through the skin is 0.5 mg/cm²-hr (McLaughlin, 1984).
- 100 percent of the compound is absorbed through the skin.
- An individual swims 2.6 hrs per day, 7 days per year (Versar, 1984).
- An average lifetime is 70 years.

A dose associated with dermal absorption of the maximum concentration of methylene chloride (854 µg/l) in Deepavaal Brook can be calculated as follows:

Dose =
$$854 \mu g/l \times 0.5 mg/cm^2-hr \times 2.6 hours/day \times 490 days \times \frac{1 l/1,000 cm^3 \times 1 cm^3/g \times 1 g/10^6 ug \times 7,700 cm^2}{10 kg \times 70 years \times 365 days/year}$$

 $= 1.6 \times 10^{-5} \, \text{mg/kg-day}$

Risk = (q)* (dose)
where:
$$q^* = 7.5 \times 10^{-3} \text{ (mg/kg-day)}^{-1} \text{ (Table E-1)}$$

Risk = $[7.5 \times 10^{-3} \text{ (mg/kg-day)}^{-1}][1.6 \times 10^{-5} \text{ mg/kg-day}] = 1.2 \times 10^{-7}$

G.2.6 Accidental Ingestion During Swimming

Assumptions used to estimate the dose associated with accidental ingestion during swimming include:

- An individual ingests 50 ml during swimming (Cabelli, 1983).
- The average weight of a child is 10 kg (Versar, 1984).
- An individual swims 7 days/year.
- An expected lifetime is 70 years.

A dose associated with accidental ingestion of the maximum concentration of methylene chloride (854 $\mu g/I$) detected in Deepavaal Brook can be calculated as follows:

Dose =
$$\frac{854 \text{ } \mu\text{g/l} \times 50 \text{ ml/day} \times 1 \text{ } l/1,000 \text{ ml} \times 7 \text{ } \text{days/year} \times \frac{70 \text{ } \text{years} \times 1 \text{ } \text{mg/}10^3 \text{ } \text{ug}}{14 \text{ kg} \times 70 \text{ } \text{years} \times 365 \text{ } \text{days/year}}$$
= $8.1 \times 10^{-5} \text{ mg/kg-day}$

Risk = $(q)^* \text{ (dose)}$

where: $q^* = 7.5 \times 10^{-3} \text{ (mg/kg-day)}^{-1} \text{ (Table E-1)}$

Risk = $[7.5 \times 10^{-3} \text{ (mg/kg-day)}^{-1}][8.1 \times 10^{-5} \text{ mg/kg-day}] = 6.1 \times 10^{-7}$

G.2.7 Ingestion of Fish

Assumptions used to estimate the dose associated with ingestion of fish include:

- An individual ingestion 6.5 g of fish per day.
- An adult weights 70 kg.
- An expected lifetime is 70 years.

To calculate the bioconcentration factor, the following equation is used (Lyman et al., 1982)

$$\log$$
 BCF = 0.76 \log Kow - 0.23

where: Kow = octanol/water partition coefficient (Table 8-1)

Dose associated with ingestion of fish from Deepavaal Brook, which contains a maximum concentration of methylene of 854 µg/l, can be calculated as follows:

$$\log$$
 BCF = 0.76 (1.25) - 0.23

log BCF = 0.72

BCF = 5.24

Dose =
$$\frac{854 \text{ } \mu\text{g/l} \times 5.24 \times 6.5 \text{ g/day} \times \text{mg/}10^3 \text{ } \mu\text{g} \times \text{kg/}10^3 \text{ g}}{70 \text{ kg}}$$

$$= 4.1 \times 10^{-4} \text{ mg/kg-day}$$

Risk =
$$(q)*(dose)$$

where:
$$q^* = 7.5 \times 10^{-3} (mg/kg-day)^{-1} (Table E-1)$$

Risk =
$$[7.5 \times 10^{-3} (mg/kg-day)^{-1}][4.1 \times 10^{-4} mg/kg-day] = 3.1 \times 10^{-6}$$

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